ON PARAPLEGIA FROM POTT'S DISEASE.[1]

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IN this short paper I wish to draw attention to some points in the pathology and treatment of paraplegia induced by Pott's disease of the vertebræ which appear to me of physiological interest, and also of some practical importance; and in order to give, as it were, chapter and verse for what I am going to say, I will begin with a short account of a case of this disease which was some months ago under my care at the hospital, and which will serve to illustrate the principal points upon which I desire to dwell. This was the case of a florid-looking girl, aged 20, who had been in good health during childhood, and had not suffered from any manifestation of the scrofulous diathesis in the glands or other parts, nor had there been any phthisis in the family of the patient. There was no history of injury to the spine. She had been in domestic service until two years ago, when she had rheumatic fever, which confined her to bed for six weeks. Soon afterwards, however, she was well enough to take another situation, in which she remained for nine months. She then, apparently without any cause, began to feel pain in the right foot, which presently became very severe, and was followed by loss of power in the right leg. This disabled her from attending to her duties. Eventually the left leg was likewise affected by pain and paralysis, and the patient was then confined to bed, being

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unable to sit, walk, or stand. She was admitted into a provincial hospital, where she remained under treatment for four months, but at the end of that time her condition had not improved, except that the pain was lessened.

On examination, I found that there was almost complete paralysis of the right lower extremity, from the hip downwards, the patient being only able to impress a very slight movement to the hip-joint. The muscles were flabby, but apparently not wasted. The faradic and galvanic responses of the nerves and muscles were obtained with great facility and an unusually low current strength; and there were no qualitative changes, the cathode closing contraction appearing before the anode opening contraction, and cathode closing tetanus being easily induced. The superficial reflexes were likewise unusually brisk, as pricking or tickling the sole or any other part of the limb excited considerable jactitations, not only of the right, but also of the left leg. The same was the case with the deep reflexes, for the limb was spasmodically thrown about, not only on tapping the ligamentum patellæ, but any point of the belly of the rectus femoris, the tibia, etc. Ankle clonus was easily excited, and continued for several minutes. There was, however, no tendency to spontaneous cramps or jactitations, and no muscular rigidity anywhere.

The foot was habitually cold and clammy, and occasionally quite wet; the coldness was rather less in the leg, and the thigh was tolerably warm. Round the ankle and on the instep there were large livid patches which disappeared on pressure. There was, however, not the peculiar appearance of glossy skin. The different forms of sensation in the entire limb were perfectly normal, there being neither analgesia nor anæsthesia to touch, pressure, or heat and cold.

The *left* lower extemity was in all respects less affected than the right. The patient could bend the knee and draw the leg a little way up, and had also some little power in the foot and toes; but all these movements were very feeble, and she was quite unable to stand on

the leg. The superficial and deep reflexes were exaggerated, but not to the same extent as in the right limb; and a higher degree of faradic and galvanic power was required for eliciting good responses of the nerves and muscles. The vaso-motor symptoms of coldness, etc., about the ankle and instep were likewise less developed; and sensibility in all its forms was perfectly normal.

Examination of the spine revealed an angular deformity of the ninth and tenth dorsal vertebræ, the spinous processes being prominent in the centre, while the transverse and oblique processes protruded laterally. The structures above the projecting vertebræ appeared to be thickened and indurated; but the swelling was not painful, nor tender on pressure or percussion. There had never been any pain in the spine. No fluctuation could be made out, and there was no sign of an abscess, either in the spinal region or in the psoas muscle, or elsewhere. There was no increase of temperature in the swelling; but the flexibility of the spine was much diminished. The commencement of the deformity had coincided with the appearance of the pain and paralysis in the right leg fifteen months ago.

There was no affection of the bladder and rectum, no tendency to bed-sores, and the catamenia appeared regularly every two months. All the other organs and functions of the body were normal.

Under these circumstances, the diagnosis could not be doubtful. We had evidently to do with caries, probably non-tubercular, of the bodies of two of the dorsal vertebræ, with their intervertebral cartilages and accessory ligaments; while the contact of the diseased structures with the dura mater had led to an inflammation in the external layers of that membrane, which had resulted in thickening and the formation of cheesy products pressing on the spinal cord. In the latter, there was probably some degree of interstitial myelitis set up, more especially in the crossed pyramidal strands, as shown by paralysis and increase of deep reflexes, and also in the posterior columns, as shown by the severe neuralgic pain in the feet, of which

the patient had complained more especially in the beginning of the affection; while the central gray matter of the cord had probably remained normal.

The patient was at first treated with large doses of iodide of potassium and cod-liver oil, and friction of the paralyzed limbs with ammonia liniment. The result of this treatment, however, which was carried on for nearly three months, was absolutely *nil* so far as progress towards recovery was concerned, although it may have checked the further progress of the disease. I then requested Mr. Pearce Gould to apply the actual cautery to the spine on both sides of the deformity; and this was very thoroughly and effectually done by him on four separate occasions. After the second application there was a decided improvement in the paralysis, and after the fourth the patient had so far recovered that she was able to walk about as usual. The vaso-motor symptoms of coldness and lividity, and the excessive perspiration in the feet, had then disappeared, and the superficial and deep reflexes were almost normal, although still somewhat exaggerated. The patient was kept under observation for another six weeks, and as she continued well, was discharged from the hospital and sent to a convalescent home.

Patients suffering from paraplegia owing to Pott's disease have sometimes recovered by the opening of abscesses either near the seat of the disease or in the psoas muscle, or elsewhere. In the present case, however, there had never been any sign of an abscess; and, as the patient had been treated for a considerable time, both in the country and in London, by rest in the recumbent position and other measures without any improvement in the paralysis, we cannot doubt that it was the powerful counter-irritation set up by the use of the actual cautery which was really the curative agent in the present instance; for soon after it had been applied improvement was noticed, and this went on steadily to a cure under repeated applications of the same agent. We must therefore assume that under its influence the inflammation in the bones and the dura mater was

arrested, that there was absorption of morbid products which had been deposited in the external layers of the membrane, whereby the compression of the cord was relieved, and the interstitial myelitis which had been set up in several of the white columns of that organ was likewise cured.

The first symptom in the present case was pain in the right foot, which came on suddenly and was exceedingly severe; on the other hand, there had never been any pain in the back. The pain in the foot, which, according to the description given of it by the patient, appears to have resembled the lightning pains of locomotor ataxy, was evidently caused by the inflammation which then commenced in the dura mater and the cord, as it almost coincided with the beginning of the paralysis. Was the pain in the foot owing to irritation of the nerve-roots in their exit through the inflamed membrane, or to irritation of the membrane itself, or to myelitis? As the inflammation of the dura mater in the present case was most probably confined to its anterior aspect, where the anterior or motor roots emerge through the membrane, it is not likely that it was owing to irritation of these roots, which would have produced spasm rather than pain. Was it then owing to inflammation of the dura? Here it is worthy of remark that, although Kölliker has found no nerves in that membrane, Ruedinger has been able to trace them; and Vulpian has experimentally shown that the dura, although only slightly sensitive in health, may become highly sensitive when irritated or inflamed. Indeed the membrane only needs exposure to the atmosphere for a few hours to become exceedingly irritable; and when an irritant substance is thus applied to it, the animal yells with pain, while the pupils are dilated each time that the membrane is touched. The pupil being probably the most delicate æsthesiometer, any dilatation of it under the influence of an irritation of a tissue, shows that the latter is sensitive. I do not think, however, that the pain in the present case was owing to inflammation of the dura, which would no doubt have caused local pain and tenderness; and I am therefore in-

clined to attribute the violent excentric pain of which the patient has complained to irritation of the posterior columns of the cord.

The symptoms of coldness, lividity, and excessive perspiration about the ankles and insteps showed that there was some degree of vaso-motor as well as secretory paralysis combined with spasm of the arterioles. Vaso-constrictor as well as vaso-dilator fibres are known to pass through each segment of the spinal cord and run, most probably, in the lateral columns of the organ. The co-existence of paralysis in the present case, owing, as I shall presently show, to disease of a portion of the lateral columns, thus renders the interference with vaso-motor function which was observed easily intelligible. Patches of venous congestion have the same significance as excessive perspiration; for Claude Bernard has shown that irritation of the fibres supplying the sweat-glands causes arrest of secretion, while paralysis of these fibres leads to excessive sweating. Systematic rubbing of the legs and feet with stimulating liniments did nothing whatever to modify this condition, thus plainly showing that it was caused by central disease.

It was formerly believed that the paralysis which is so frequently seen in Pott's disease was owing to softening and actual destruction of the spinal cord. This view, however, seemed to be inconsistent with the recovery of power, which sometimes occurs even without active treatment. In order to reconcile this apparent discrepancy, Mr. Shaw has suggested that, after the portion of the cord corresponding to the diseased vertebræ has been destroyed, the continued bending down of the column into an angle allowed the sound parts above and below to come into contact and displace the diseased part, so as to unite and be able to resume their function. He also believed another ground for entertaining hopes of recovery to be, that the loss of substance in the cord was usually confined to the anterior columns; and as these constituted only a small portion of the cord, they would be more easily replaced by healthy structures. Both these theories appear to me extremely hazardous, and not in consonance

with the teaching of modern physiology and morbid anatomy. A portion of the cord which has once been actually destroyed can never again act as a centre or path for voluntary power, nor can we suppose that such a loss could be replaced, even at best, by anything else but connective tissue. Moreover, it is not at all the fact that only the anterior columns of the cord suffer from the compression. Indeed, if it were so, it is unlikely that paraplegia would result; for the crossed pyramidal strands, which we know to constitute the motor paths in the cord, do not course in the anterior portion of that organ, but in the posterior part of the lateral column close to the posterior gray cornua.

Another important and suggestive fact is, that a similar form of paralysis as in Pott's disease is caused by pressure from hydatid cysts, aneurism, abscess, or malignant disease, invading the organ from behind or laterally. It is, therefore, far more rational to assume that the entire cord suffers some degree of compression in paraplegia from Pott's disease.

That simple pressure on the cord may cause paraplegia has been experimentally shown by Vulpian, who introduced a small bit of a wooden match under the arches of the lower dorsal vertebræ in a guinea pig, and thereby caused paralysis in the hind legs, but without loss of sensibility or reflex motility. A quarter of an hour afterwards the bit of wood was withdrawn, and in another hour it was found that the hind limbs had recovered their motive power. It is, however, not in this simple way that paraplegia is produced in Pott's disease. Echeverria, Michaud, Charcot, and others have, on the contrary, shown that there is habitually inflammation of that portion of the dura mater, more particularly in its external layers, which corresponds to diseased vertebræ, and that this inflammation may spread both upwards and downwards beyond the seat of the caries. It is frequently confined to the anterior portion of the dura, but may extend through its whole circumference. The arachnoid and pia mater are frequently healthy, but interstitial myelitis seems always

to be present, and may affect, not only the white columns, but also the gray matter; and it is in severe cases followed by secondary degeneration of the crossed pyramidal strands below the seat of the lesion, and by ascending degeneration of the postero-internal or Goll's columns, which latter is chiefly seen in the cervical portion of the cord. There could be no doubt that in the present case the crossed pyramidal strands were affected, either by interstitial myelitis or by a degree of secondary degeneration—more probably the former, as shown by the symptoms of paralysis combined with exaggeration of the deep reflexes; and this myelitis was evidently more severe in the right than in the left half of the cord, as the two symptoms just mentioned were much more marked in the right than in the left lower extremity.

How have we to explain the fact that, while there was almost complete motor paralysis, at least in the right limb, yet there was no loss of sensibility? This is a difficult question which has not as yet received a satisfactory answer, and which I will now proceed to consider.

Taking the clinical symptoms of Pott's disease in its different aspects as a guide, it appears to me that we may distinguish *three different degrees of pressure* and subsequent morbid changes as far as the cord and the spinal membranes are concerned. We meet with cases of Pott's disease in practice where there is considerable deformity, but no paralysis, or any other nervous symptoms referable to the spine. In such cases I believe that the pressure which no doubt exists is only just sufficient to displace the spinal liquid in which the cord floats, but leaves the membranes and the substance of the cord itself intact.

The second degree of pressure I would assume to be that which exists in the case which I have just related, and where we have to do, not only with displacement of spinal liquid, but also with a moderate amount of external pachymeningitis and interstitial myelitis, causing paraplegia, but no loss of sensibility or reflex motility, and affecting therefore chiefly the white columns, but not the central gray matter. The posterior columns are not by any

means the only channels by which sentient impressions are conveyed, and sensibility will persist as long as a small portion of the nerve cells in the gray centre of the cord remains active. This fact was demonstrated by Schiff and Brown-Séquard. It has been suggested by Vulpian that white nerve-tubes are more easily compressed than gray cells; but, although this appears possible, it cannot be said to have been proved. There can, however, be no question that motion suffers more readily from pressure and other morbid influences than sensation; and that, where both functions have suffered in consequence of disease of the nervous centres or peripheral nerves, sensation is more apt to return, and more liable to be re-established by therapeutical procedures, than motion. In that form of paralysis of the musculo-spiral nerve which is clinically seen after contusion of that nerve, there is rarely anæsthesia, although the paralysis may be almost or quite complete. Hemi-anæsthesia from cerebral lesion is more easily recovered from than hemiplegia, owing to a similar lesion. In the last stage of tabes dorsalis, when the patient may be completely paralyzed and anæsthetic in his lower extremities, powerful faradization of the skin with a wire brush will often, at least temporarily, restore sensation. All these facts tend to show that, other things being equal, motion is more easily lost than sensation, and that the latter, even when its ordinary channels are closed, may be more readily transmitted along other channels than motion which sticks to certain hard and fast lines. This is also proved by the fact that, as far as I know, there is a not a single case of Pott's disease on record where there has been loss of sensation while the power of motion remained; while the reverse is a very ordinary occurrence.

The third degree of pressure and its consequences in Pott's disease I assume to be that where we have to do with complete transverse myelitis, affecting not only the white columns, but also the entire gray centre of the cord at the level of the disease of the vertebræ, and being then accompanied with secondary degeneration of the pyramidal strands below the lesion and ascending secondary

degeneration in Goll's column in the cervical region. The necessary consequence of this must be not only paraplegia, but also anæsthesia and analgesia in all parts below the lesion, with paralysis of the bladder and rectum, and tendency to bed-sores. A case of this kind has been recorded by Buzzard in the "Transactions of the Clinical Society" for 1880. His patient died of obstruction of the bowels, apparently brought on by the application of a Sayre's jacket, and after death the mid-dorsal region of the cord was found to be completely disorganized. It is, therefore, seen that, with regard to prognosis, *loss of sensibility in a case of Pott's disease is a far graver symptom than paralysis*, inasmuch as it shows a more severe degree and a greater extent of myelitis, which then appears to have affected the entire transverse section of the cord. We know that this condition leads not only to complete paralysis and anæsthesia in all parts below the lesion, but also to paralysis of the bladder and bowels, deep bed-sores, and blood poisoning, thus placing the life of the patient in the greatest jeopardy. It is obvious that recovery must be impossible when such severe lesions have been produced; and I would therefore recommend an early resort to the actual cautery in all cases of Pott's disease where symptoms of paraplegia are beginning to be developed.

In conclusion, I will say a few words about the use of the cautery in spinal affections, and the vicissitudes which it has undergone in the course of time in medical estimation. During the first half of the present century, this agent was quite indiscriminately used in all spinal affections of whatever nature, and patients often underwent a great amount of torture without deriving any corresponding degree of benefit from the same. Romberg was the first to object to this treatment in cases of tabes in which he had found it quite ineffective, and probably in consequence of his protest, the use of the cautery declined a good deal during the next twenty or thirty years. My experience is, that the cautery does no good in affections of the substance of the cord, such as tabes, spastic spinal paralysis, multiple sclerosis, infantile paralysis, progressive

muscular atrophy, etc.; but that it is of very considerable service in disease of the spinal membranes from whatever cause. The paraplegia of Pott's disease has recently been more especially treated with Sayre's jacket and large doses of iodide of potassium, which I believe to be inferior in efficacy to the cautery; and I shall, therefore, be much gratified if the use of the latter will now be again more generally resorted to than it has been of late years in cases of paralysis from Pott's disease.